



## Rehabilitation and Treatment Algorithm for Patients with Ocular Ischemic Syndrome on the Background of Arterial Hypertension

1. Zhalalova D. Z.

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<sup>1</sup> Samarkand State Medical Institute.

**Annotation:** Under conditions of chronic hypoxia, there are functional disorders of autoregulation of blood circulation in the vessels of the eye. One of the common pathogenetic factors is a violation of vascular autoregulation, which is extremely important to take into account in the pathogenesis of the disease.

**Keywords:** ischemic syndrome, etiopathogenetic treatment, arterial hypertension, rehabilitation.

**Purpose.** To develop an algorithm for the treatment of patients with ocular ischemic syndrome (OIS) against the background of arterial hypertension and their rehabilitation.

**Material and Methods.** Under observation were 11 patients diagnosed with GIS, including 7 men and 4 women. To treat this pathology, etiopathogenetic therapy was used in conjunction with neuroprotective treatment. Depending on the method of treatment, the patients were divided into 2 groups: group 1 — 5 patients, 2 of them with a diagnosis of ischemic central retinal vein thrombosis (CRV) and 3 with a diagnosis of anterior ischemic neuropathy (AID). group 2 — 6 patients, 3 of them with a diagnosis of ischemic thrombosis of the CVD and 3 with a diagnosis of IDU. In group 1, patients received etiopathogenetic treatment, in group 2 — etiopathogenetic treatment + neuroprotective therapy.

Treatment of patients of group 1 with a diagnosis of ischemic thrombosis of the CVS was as follows: losartan 50 mg, diacarb 0.25 mg 1 tab. 2 times a day, Panangin 1 tab. 3 times a day. Arutimol 0.5% solution, 2 drops 2 times a day. Fraxiparine 0.15 and dexamethasone solution 1 ml were administered paravasally, intravenously — trental 100 mg/ day drip, 5 infusions every other day; inside - 300 mg per day for 2 months, rheosorbilact. Sulodexide intramuscularly 600 LPL U 10 injections, then - orally 250 LPL U 2 times a day for 30-40 days, thrombo ACC 100 mg per day once for 6 months. and more, angioprotector - dicynone and anti-sclerotic drugs.

Patients with a diagnosis of group 1 IDUs received the following treatment: locally 0.4% dexamethasone solution + dicynone parabolbarno 0.5 ml, fraxiparine 0.15. Inside Diakarb 0.25 mg 1 tab. 2 times a day, Panangin 1 tab. 2 times a day, intravenously rheosorbilact drip 200 ml 1 time a day, arutimol 0.5% solution 1 drop 2 times a day. paravasal fraxiparine 0.15. In group 2, neuroprotective treatment was additionally applied to the above therapy. Gliatilin intravenously at a dose of 1 g 1 time

per day for 5 days, followed by 1 table. 2 times a day for 1 month; Cerebrolysin was administered paravasally at a dose of 0.5 ml once a day for 7 days, then the course was continued in the form of intramuscular injections of 5 ml for up to 21 days.

In group 1 with a diagnosis of CVD thrombosis, the visual acuity (VA) of patients before treatment was  $0.06 \pm 0.01$ , in patients with PIN, vision was  $0.03 \pm 0.01$ . In group 2 with CVD thrombosis, the VA of patients before treatment was  $0.08 \pm 0.02$ ; in patients with PIN, the VA was  $0.02 \pm 0.01$ . The field of view of patients in group 1 with CVD thrombosis before treatment was  $365 \pm 25^\circ$ , with PIN — prolapse of the lower part with residual vision on one side. In group 2 with CVD thrombosis, the field of view was  $345 \pm 34.5^\circ$ , with PIN — loss of 2/3 of the field of view. In the fundus of the eye in patients of both groups with CVD thrombosis, the changes were identical: edema of the optic disc was pronounced, the arteries were sharply narrowed, the veins were dilated, tortuous, and dark. Multiple hemorrhages were noted. Ophthalmoscopy for PIN: pale edematous optic disc, wide, dark, tortuous veins. Small hemorrhages were observed on the disc and in the peripapillary zone. Disk prominence and hemorrhages along the small veins were noted.

### Results.

After treatment in group 1 with CVD thrombosis, visual acuity increased to  $0.1 \pm 0.05$ , in patients with PIN, vision was  $0.09 \pm 0.02$ . In group 2 with CVD thrombosis, the VA of patients after treatment was  $0.3 \pm 0.05$ ; in patients with PIN, the VA increased to  $0.2 \pm 0.05$ . The field of view of patients in group 1 with CV thrombosis after treatment was  $395 \pm 27.5^\circ$ , with PIN the prolapse of the lower part decreased by  $15^\circ$ . In the 2nd group with CVD thrombosis, the visual field was  $415 \pm 38.5^\circ$ , with PIN, visual field loss decreased by  $25^\circ$ .

In the fundus of patients of group 1 with CV thrombosis, the changes were as follows: edema of the optic disc slightly decreased, arteries were narrowed, veins were slightly dilated. Hemorrhages have decreased somewhat. Ophthalmoscopy in group 1 PIN: edema of the optic disc decreased, veins were dilated. On the disc and in the peripapillary zone, small hemorrhages slightly decreased.

The fundus of the eye in patients of the 2nd group with CVD thrombosis: the edema of the optic disc decreased, the arteries were slightly narrowed, the veins were slightly dilated. Ophthalmoscopy of patients with PIN of the 2nd group: edema of the optic disc decreased, veins were dilated. On the disc and in the peripapillary zone, small hemorrhages decreased.

### Conclusion.

The results of the treatment showed that the complex therapy of ocular ischemic syndrome, including neuroprotectors and nootropics, has a positive effect on visual functions.

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